therapy, they had their biomarkers evaluated. Identification of silent cardiac TOD was done by transthoracic echocardiography, stress echocardiography, and/or myocardial perfusion imaging. Carotid–femoral pulse wave velocity.

Results: Results showed that 96 (35%) patients had evidence of cTOD. Left ventricular hypertrophy evaluated by LV mass index showed the highest prevalence (32.7%), followed by left ventricular diastolic dysfunction (28.9%), left atrial enlargement (19.1%), systolic dysfunction (10.6%), ischemia (7.1%) and the lowest was PWV (2.7%). The discrimination power as evaluated by area under the curve [AUC] for BNP to identify any form of silent cTOD was 0.79 overall and 0.83 in men, while for hs-cTnT it was 0.70 and 0.74 in women. The combined AUC for BNP and hs-cTnT together was 0.81 and 0.82 in men. Week discrimination power existed for of other biomarkers, with AUCs of 0.61 for microalbuminuria,, 0.60 for hs-CRP, and 0.58 for eGFR.

Conclusions: Asymptomatic patients treated for primary prevention, existing silent cTOD could be identified by BNP screening. The results of hs-cTnT was weaker than that of BNP. Combining BNP plus hs-cTnT together showed best results. Primary prevention could be improved by Prescreening with BNP ± cTnT followed by phenotyping.

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Effect of circadian rhythm of blood pressure on arterial wall stiffness and on left ventricular diastolic dysfunction

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Background: Arterial stiffness is a risk factor for cardiovascular morbidity and mortality. Variability of blood pressure has been reported to be related to worse cardiovascular outcome. The relationship between the arterial stiffness and the circadian rhythm of blood pressure (BP) has been controversial. The objective was to the examine impacts of BP variability on left ventricular diastolic function and arterial stiffness in the hypertensive patients.

Methods: Ambulatory BP monitoring, pulse wave velocity, and echocardiography were performed in 268 patients (153 males, 47 ± 11 years) with HTN and pre-HTN. BP was measured at the outpatient clinic and 24-h ABPM was performed. Using carotid femoral applanation tonometry, PWA was performed for evaluation of systemic arterial stiffness expressed as augmentation index. Echocardiograms were performed and an average of 3–6 cardiac cycles was done for all measurements including the left ventricular mass index (LVMI), relative wall thickness (RWT), trans mitral flow propagation velocity (Vp), TDI, and midwall shortening fraction (MWSF). Nocturnal dipping was defined as a reduction of diastolic BP (DBP) by >10% of systolic BP (SBP) when compared with the daytime values. Isolated systolic non-dipping, is reduction of <10% in the SBP, When compared with the daytime values. Isolated diastolic non-dipping is reduction of <10% in the DBP. Both systolic and diastolic non-dipping is reduction of <10% in both SBP and DBP.

Results: Among groups, the clinic SBP and DBP, daytime mean BP of 24-h ABPM, gender and body mass index were not statistically different. Augmentation pressure (AP), augmentation index (AI) showed statistically significant difference ($p = 0.008$ and 0.021, respectively). Multivariate analysis showed that isolated diastolic non-dipping was correlated with arterial stiffness expressed as AI only in young group.

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Feasibility of using ivabridine in adult congenital heart disease patients

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Background: Beta Blockers have been the main agent to control the heart rate in Fontan patients. Ivabridine which is a selective SA node inhibitor has been used in adult cardiac patient with successful result in controlling heart rate for those who did not respond to beta blockers alone. This agent has not been reported to be used in adult congenital heart disease patients.

Objective: To determine the feasibility of using ivabridine in patient with adult congenital heart disease and inappropriate sinus tachycardia who failed to respond to beta blockers.

Methods: 3 patients all with single ventricle physiology were on maximum tolerable dose of Beta Blockers, with inappropriate sinus tachycardia started on ivabridine. Average follow up was 52 weeks with regular holter monitor. Pro-BNP, 6 min walk test, and echocardiography at baseline and 24 weeks of follow up. Physical examination and interrogation for any detected side effect was done on each visit.

Result: At baseline the average heart rate was 100 ± 4 BPM, ivabridine started on with target dose 7.5 mg BD. The mean average heart rate at 12 weeks was 83 BPM, 78 BPM on 24 weeks, and 85 BPM on 52 weeks. There was no reported side effect by the patients or an effect on single ventricle function.

Conclusion: ivabridine can be used in single ventricle patients to reduce the heart rate in conjunction with beta blockers. However larger study is needed to assess its isolated long term effect on a diseased sinus node.

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Identification of a novel homozygous NEXN gene mutation in recessively inherited dilated cardiomyopathy